Genotype by Environment Interaction and Genetic Correlations Among Parities for Somatic Cell Count and Milk Yield

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ABSTRACT

Lactation measures of somatic cell concentration and total SCC production were developed. Data were separated into three parity groups. Within parity, five data sets were created: four subsets by herd-year average SCC, and one with all records. Records on lactation SCC, total SCC production, and 305-d milk were analyzed by a sire model separately in each subset within parity. Variance components estimates were by REML. For SCC and total SCC production, heritability estimates averaged .12 and were lowest in the highest level of herd-year average SCC. Estimates of genetic correlation between SCC and total SCC production were over .95; between SCC and 305-d milk were around .25 in first and −.15 in later parities; between total SCC and 305-d milk were around .50 in first and .15 in later parities. Product-moment correlations between sire effects in different levels of herd-year average SCC were obtained. Ratios of product-moment correlations to their expected value were above .80 for all traits in all parities. High ratios indicated little genotype by environment interaction. A sire by herd interaction was fitted in the model and accounted for less than 2% of total phenotypic variance for SCC and total SCC production, and 4% for 305-d milk. Estimates of genetic correlation of first with later parities were .71 to .86 for all traits. Between second and third parity genetic correlation estimates were around unity for all traits. Records from all parities should be used for sire evaluation. (Key words: genotype environment interaction, genetic correlation, somatic cell count)

INTRODUCTION

Genetic variation in mastitis incidence in dairy cattle has been documented (13). Milk SCC has been presented as an accurate indicator of mastitis and a useful criterion for selection decisions (4). Genetic correlations between SCC variables and measures of mastitis occurrence have been estimated in several studies (4, 7, 9) and were, on the average, moderately high and positive. Further, frequency of clinical mastitis and probability of treatment increase with SCC (5). Grootenhuis (8) compared daughter groups and found heifers with low SCC had older half sisters with lower rates of infection than heifers with high SCC. Also, sire progeny groups with low average SCC in first parity had lower rates of mastitis and lower SCC in later parities than groups with high average SCC in first parity (29). Finally, positive correlations between sire evaluation for SCC and daughter average infection rates have been reported (4). Conclusions from these reports suggest that genetic evaluation and selection against SCC would result in reduction of mastitis incidence.

Heritability estimates and measures of SCC are moderately low, according to several reports (7, 9, 11, 23). Hence, any response to selection would be slow, yet permanent. Lactation measures of SCC appear to have higher heritability than test-day observations (9, 22). In a simulation study, Strandberg and Shook (25) found that breeding programs that include mastitis or SCC could diminish the rate of increase in mastitis that accompanies genetic

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improvement for milk yield. Selection can be effective when applied against sires whose daughters have high milk SCC, indicating frequent mastitis infections. Hence, estimates of sires' breeding values for SCC are needed.

In support of sire evaluation and selection research on the consistency of genetic parameters and sire effects across environments, i.e., by environment interaction (G×E), is required. Presence of G×E in dairy cattle has been researched for milk and milk components (26, for a review). This interaction has been evaluated from estimates of correlations between sire effects in different environments (6), or from the variance due to sire by herd interaction (S×H) (17, 27). No previous work has been reported on G×E for milk SCC. The objectives of the present study were: 1) to investigate the consistency of sire effects and genetic parameters of two SCC lactation traits and lactation milk yield across levels of herd average SCC within parity; 2) to estimate the variance due to S×H for the SCC traits and milk; and 3) to evaluate genetic correlations among parities for these traits.

MATERIALS AND METHODS

Monthly test-day milk yield and SCC, and lactation milk yield records on 972,799 Holstein cows were provided by the Wisconsin DHIA between 1978 and 1987. Only records from first, second, and third parity, and lactations with at least 1 test-day observation in the first 65 days and 5 tests in 305 d were kept. Additional observations were eliminated if sire registration number, milk yield, or age at calving were missing, if sire was not Holstein, or if lactation was shorter than 230 d, leaving records on 101,006 first, 71,627 second, and 48,606 third parity cows.

Two test-day SCC measures were considered: 1) a measure of SCC concentration

$$SCS = \log_2\left(\frac{TSC}{100}\right) + 3$$

2) and a measure of total SCC production

$$SCT = \ln (TSC \times TML)$$

where TSC was test-day, SCC was in cells per microliter, and TML was test-day milk yield in kilograms. With logarithmic transformation, normality and homogeneity of variances were achieved (1). Test-day SCS and SCT were adjusted for stage of lactation and calendar month effects, within parity, by a procedure similar to that of Wiggans and Shook (30). Adjusted test-day records were averaged to form two lactation traits: SC was the average of SCS and total SCC production (LT) was the average of SCT. Lactation milk yield (ML) in 305 d unadjusted for age and season of calving was included in the analysis.

Within parity, five data sets were created: four subsets of approximately equal size by herd-year average SC (HAVSC) and one with all records. Each herd-year subclass was required to have at least 4 records in first, 3 records in second, and 2 records in third parity. Furthermore, because genetic parameters and sire effects were of interest across levels of HAVSC, each sire was required to have daughters with records in at least two herds in each of four subsets within parity set. The numbers of records, herds, herd-year-seasons, and sires by level of HAVSC within parity, are in Table 1.

Two models of analysis were considered for each data set. They can be expressed in the following general form:

$$Y_{ijklm} = HYS_{ij} + b_1X_{ijklm} + b_2X^2_{ijklm} + G_k + S_{kl} + C_{ikl} + e_{ijklm}$$

where HYS_{ij} is the fixed effect of year-season of calving j in herd i; X_{ijklm} is age at calving; b_1 and b_2 are linear and quadratic regression coefficients on age at calving; G_k is the fixed effect of sire group k based on sire birth year, k = 1,. . .,4; S_{kl} is the effect of sire l in group k randomly distributed around zero with variance $A\sigma^2_s$, where A is the numerator relationship matrix between the sires due to their sires, and e_{ijklm} is the residual randomly distributed around 0 with variance $I\sigma^2_e$, where I is the identity matrix. Under Model [1], $C_{ikl} = 0$. Variance components were estimated by REML using the algorithm described by Meyer (16). Genetic correlations between traits were estimated using an algorithm that performs a canonical transformation of the traits, thus reducing a multitrait problem into a series of single-trait analyses (15). Under Model [2], C_{ikl} was

TABLE 1. Number of records, herds, herd-year-seasons (HYS), and sires by level of herd average SCC (HAVSC) within parity.

HAVSC	Records	Herds	HYS	Sires
First parity				
Q1 ≤ 2.30	20,571	1267	4038	691
Q2 2.31 to 2.64	20,040	1315	3709	691
Q3 2.65 to 3.02	19,868	1401	3879	691
Q4 > 3.02	19,590	1413	4298	691
Total	80,069	3016	15,924	691
Second parity				
Q1 ≤ 2.37	14,535	1253	3799	624
Q2 2.38 to 2.81	14,091	1274	3556	624
Q3 2.82 to 3.28	14,096	1358	3741	624
Q4 > 3.28	13,955	1424	4209	624
Total	56,677	3019	15,305	624
Third parity				
Q1 ≤ 2.64	10,008	1338	3742	534
Q2 2.65 to 3.14	9818	1298	3345	534
Q3 3.15 to 3.64	9833	1362	3493	534
Q4 > 3.64	9703	1578	4121	534
Total	39,362	3155	14,701	534

the interaction between sire kl and herd i randomly distributed around zero with variance $I\sigma^2_{sh}$. Although sires were related, S×H effects were assumed independently distributed due to computing limitations. Variance components were estimated by REML using an algorithm for univariate analysis described by Smith and Graser (24).

Product-moment correlations were calculated between sire effects in different levels of HAVSC under Model [1]. These were computed for each trait within parity. The ratio of observed to expected correlation is an estimate of the correlation of expression of the same genotype in two environments (2). In absence of G×E, the expected correlation of sire effects in two different environments, adapted from Hickman et al. (10), is:

$$r = \frac{\sum_{i=1}^{ns} R^2_{1i} R^2_{2i}}{\sqrt{\sum_{i=1}^{ns} R^2_{1i} \sum_{i=1}^{ns} R^2_{2i}}} \quad [1]$$

where r is the expected value of the correlation coefficient between sire effects, ns is the number of sires with evaluations in two different environments, and R^2_{1i} and R^2_{2i} are the squared correlations between true and estimated transmitting ability of sire i in environments 1 and 2. R^2_{1i} and R^2_{2i} are also reliabilities of sires' solutions. Reliability can be expressed as:

$$R^2_i = 1 - \frac{PEV_i}{\sigma^2_s} \quad [2]$$

where PEV_i is the prediction error variance association with sire i. Approximate estimates of PEV were obtained using the effective number of daughters of the sires, since the latter was the only pertinent information available. The PEV can be estimated as:

$$PEV_i = (\sigma^2_e)\ (dinv_i)$$

where $dinv_i$ is the diagonal element of the inverse of the coefficient matrix pertaining to sire i. Furthermore, $dinv_i$ can be approximated by $(diag_i)^{-1}$, where $diag_i$ is the diagonal element of the original coefficient matrix pertaining to sire i. This approximation gives the lower bound of PEV described by VanRaden and Freeman (28). Then:

$$diag_i = n_i + (\tfrac{1}{3}nsn + js)k \quad [3]$$

or

$$diag_i = n_i + k \quad [4]$$

where n_i is the effective number of daughters of sire i, nsn is the number of sons of sire i, js = 1

TABLE 2. Number of records and sires in each parity within pair of parities.

Pair of parities	Parity	Records	Sires
1 and 2	1	74,065	853
	2	46,829	853
1 and 3	1	79,791	783
	3	35,599	783
2 and 3	2	52,384	783
	3	31,913	783

if sire i has unknown sire and $js = \frac{4}{3}$ if sire i has known sire, and k is the ratio of residual to sire variance. Estimates of $diag_i$ from Equation [3] assume perfect information on all male relatives, underestimate dinv, and overestimate R^2. Estimates of $diag_i$ from Equation [4] ignore information on male relatives, and usually overestimate dinv and underestimate R^2. These extremes were used to derive approximate upper and lower limits of R^2 in Equation [2] and of expected correlations among levels of HAVSC for sire effects in Equation [1].

Consistency of sire effects from one parity to another was investigated. Genetic correlations were computed between each pair of parities for each trait. For each pair of parities, i.e., first with second, first with third, and second with third, records were edited so each sire had daughters with records in at least two herds in each parity. If there were two records on the same cow in any pair of parities, one of then was randomly removed. This was done because sire effects were to be evaluated on two independent data sets. Number of sires and records for each data set is in Table 2. Sire effects for each parity within pair of parities were estimated using Model [1]. Product-moment correlation coefficients between sire effects in different parities were calculated for each pair, and approximate limits of their expected values were estimated from Equations [1], [2], [3], and [4]. Genetic correlations between parities were the ratios of observed to expected correlation coefficients.

RESULTS AND DISCUSSION

Phenotypic means and standard deviations for SC, LT, and ML by level of HAVSC within parity are in Table 3. Overall means for all three traits increased with parity. Within parity, SC and LT means increased with HAVSC, as expected, and ML means decreased. These trends reflect decreased milk production in

TABLE 3. Phenotypic means and standard deviations for somatic cell concentration (SC), total SCC production (LT), and 305-d milk (ML) by level of herd average SCC (HAVSC) within parity.

	Means			Standard deviations		
HAVSC[1]	SC	LT	ML (kg)	SC	LT	ML (kg)
First parity						
Q1 ≤ 2.30	2.00	7.01	6804	.93	.66	1144
Q2 2.31 to 2.64	2.48	7.32	6658	.98	.69	1138
Q3 2.65 to 3.02	2.83	7.56	6603	.98	.70	1148
Q4 > 3.02	3.38	7.90	6376	.99	.70	1162
Total	2.66	7.44	6613	1.09	.76	1158
Second parity						
Q1 ≤ 2.37	2.01	7.19	8013	.94	.65	1386
Q2 2.38 to 2.81	2.61	7.57	7759	.99	.68	1362
Q3 2.82 to 3.28	3.03	7.84	7652	1.01	.70	1361
Q4 > 3.28	3.71	8.26	7279	.99	.68	1392
Total	2.83	7.71	7680	1.16	.78	1401
Third parity						
Q1 ≤ 2.64	2.22	7.39	8502	.94	.64	1465
Q2 2.65 to 3.14	2.90	7.83	8260	.97	.67	1450
Q3 3.15 to 3.64	3.40	8.14	8095	.98	.68	1444
Q4 > 3.64	4.11	8.59	7731	.91	.63	1453
Total	3.15	7.98	8149	1.17	.79	1465

[1]The HAVSC stratification from low (Q1) to high (Q4).

TABLE 4. Heritability estimates for somatic cell concentration (SC) under a noninteraction and a sire by herd interaction model, with approximate SE, by level of herd average somatic cell concentration (HAVSC) within parity.

HAVSC[1]	Heritability (%)		
	No interaction	Interaction	SE
First parity			
Q1 ≤ 2.30	13.3	12.9	2.0
Q2 2.31 to 2.64	15.2	14.8	2.2
Q3 2.65 to 3.02	12.4	12.4	1.9
Q4 > 3.02	8.7	8.3	1.7
Total	13.1	13.8	1.2
Second parity			
Q1 ≤ 2.37	11.8	11.8	2.3
Q2 2.38 to 2.81	11.6	11.6	2.3
Q3 2.82 to 3.28	11.8	11.7	2.4
Q4 > 3.28	9.9	9.9	2.2
Total	11.7	11.7	1.3
Third parity			
Q1 ≤ 2.64	13.8	14.1	3.2
Q2 2.65 to 3.14	13.8	13.5	3.2
Q3 3.15 to 3.64	9.4	9.4	2.7
Q4 > 3.64	6.1	6.1	2.6
Total	10.1	10.8	1.5

[1]The HAVSC stratification from low (Q1) to high (Q4).

poorly managed herds with high average SCC and probably high mastitis incidence. Standard deviations within HAVSC subclass did not change with the mean for all three traits.

Heritability and Variance Components

Heritability (h^2) estimates for SC by level of HAVSC within parity are in Table 4. Estimates of sire variance are in Table 5 and estimates of residual variance are in Table 6. Estimates are presented for both noninteraction and interaction model, over all records by parity, and within parity by HAVSC. Over all records, h^2 estimates decreased in later parities (Table 4) as a result of decreasing sire variances (Table 5) and increasing residual variances (Table 6). These changes suggest that environmental factors affecting SCC may become relatively more important than additive genetic factors with advancing age. Within parity, h^2 estimates were lowest in the high level HAVSC subset (Q4). This was observed in all three parities and was due to decreased sire variance in these subsets (Table 5). There were no substantial differences between h^2 estimates in the remaining three subsets. Generally, h^2 estimates for SC agree with reported estimates (7, 9, 23).

In all three parities, estimates of sire variances for SC (Table 5) were smallest in the high HAVSC subset, but they did not differ

TABLE 5. Sire variances for somatic cell concentration (SC) under a no interaction (NINT) and a sire by herd interaction (INT) model with approximate SE (SES), and sire by herd interaction variances with approximate SE (SEI) by level of herd average somatic cell concentration (HAVSC) within parity.

HAVSC[1]	Sire variance			Interaction variance	SEI
	NINT	INT	SES		
First parity					
Q1 ≤ 2.30	.030	.029	.005	.014	.010
Q2 2.31 to 2.64	.040	.039	.006	.033	.014
Q3 2.65 to 3.02	.033	.033	.005	.008	.014
Q4 > 3.02	.022	.021	.004	.022	.014
Total	.033	.035	.003	.017	.005
Second parity					
Q1 ≤ 2.37	.028	.028	.006	.018	.016
Q2 2.38 to 2.81	.032	.032	.007	.010	.020
Q3 2.82 to 3.28	.034	.034	.007	.007	.023
Q4 > 3.28	.025	.025	.006	.012	.020
Total	.031	.031	.004	.012	.008
Third parity					
Q1 ≤ 2.64	.034	.035	.008	.009	.023
Q2 2.65 to 3.14	.040	.039	.009	.019	.033
Q3 3.15 to 3.64	.028	.028	.008	.011	.036
Q4 > 3.64	.014	.014	.006	.009	.030
Total	.027	.029	.004	.012	.012

[1]The HAVSC stratification from low (Q1) to high (Q4).

TABLE 6. Residual variances for somatic cell concentration under a no interaction and a sire by herd interaction model with approximate SE, by level of herd average somatic cell concentration (HAVSC) within parity.

HAVSC[1]	Residual variance: No interaction	Residual variance: Interaction	SE
First parity			
Q1 ≤ 2.30	.870	.858	.010
Q2 2.31 to 2.64	1.012	.982	.011
Q3 2.65 to 3.02	1.032	1.024	.011
Q4 > 3.02	.994	.974	.011
Total	.976	.960	.006
Second parity			
Q1 ≤ 2.37	.918	.901	.013
Q2 2.38 to 2.81	1.074	1.065	.015
Q3 2.82 to 3.28	1.122	1.117	.016
Q4 > 3.28	.983	.971	.014
Total	1.025	1.014	.007
Third parity			
Q1 ≤ 2.64	.953	.943	.017
Q2 2.65 to 3.14	1.116	1.099	.019
Q3 3.15 to 3.64	1.164	1.153	.021
Q4 > 3.64	.912	.903	.017
Total	1.043	1.031	.009

[1]The HAVSC stratification from low (Q1) to high (Q4).

among the remaining three subsets. Sire variances over all records were within the mid range of estimates in each subset, in each parity. Genetic standard deviations for SC were estimated as twice the standard deviation between sires. These estimates ranged from .26 to .40 and are in agreement with estimates reviewed by Shook (22). In all three parities, estimates of residual variance of SC (Table 6) were larger in the intermediate HAVSC subsets (Q2 and Q3) than the extreme subsets (Q1 and Q4).

Variance component and h^2 estimates for ML are in Table 7. Estimates are over all records for each parity and under both models. Estimates of h^2 decreased with advancing parity due to increased residual variances. Sire variances did not change by parity. Within parity across levels of HAVSC, variance component and h^2 estimates for ML (not shown) fluctuated without trend, exhibiting no difference between subsets with low vs. high SCC.

Genotype by Environment Interaction

Variance components for S×H estimated under Model [2] for SC, across levels of HAVSC, within parity are in Table 5. Estimates may be somewhat biased downwards, because of the assumption $Var(S\times H) = I\sigma^2_{sh}$ in a model with related sires. This bias, however, should not be large, because relationships were due only to sires of sires, and the proportion of the relationship matrix filled was .4 to .55%. Interaction amounted to 49, 39, and 41% of the respective sire variance, over all first, second, and third parity records. Within parity by level of HAVSC, there was variation in estimates of S×H variance, but no trend was apparent due to relatively large standard errors. Although residual variances for SC consistently decreased after fitting a S×H (Table 6), sire variances re-

TABLE 7. Sire variance, residual variance, and heritability for 305-d milk (ML) under a no interaction and a sire by herd interaction model and sire by herd interaction (S×H) variance with approximate SE by parity.

	First parity	Second parity	Third parity
Sire variance, kg^2			
No interaction	54,940	57,537	57,002
Interaction	54,487	56,453	55,931
SE	4448	5770	6977
Residual variance, kg^2			
No interaction	792,749	1,180,532	1,267,931
Interaction	778,780	1,157,311	1,232,632
SE	4433	8217	11,429
S×H Variance, kg^2	15,575	26,155	39,814
SE	4075	8644	14,447
Heritability, %			
No interaction	25.9	18.6	17.2
Interaction	25.6	18.2	16.8
SE	2.0	1.8	2.0

TABLE 8. Percentage of total phenotypic variance due to sire by herd interaction for somatic cell concentration (SC) and 305-d milk (ML) by parity.

	SC	ML
First parity	1.68 (.5)[1]	1.84 (.5)
Second parity	1.14 (.7)	2.11 (.7)
Third parity	1.12 (1.1)	3.00 (1.1)

[1]Approximate standard errors in parentheses.

mained fairly constant, and they were, in some cases, larger than the estimates ignoring interaction (Table 5). These changes, however, were not large and exhibited no trend. Heritability estimates for SC (Table 4) compared between the two models followed the fluctuations of sire variance. Estimates of S×H variance over all records within parity were in the mid-range of estimates for each subset (Table 5). This suggests lack of interaction for sire by level of HAVSC. Presence of such interaction would inflate estimates of S×H over all records in comparison to estimates within subset.

Variance components for S×H for ML are in Table 7. Interaction amounted to 28% of the respective sire variance, over all first parity records. In second and third parity this value increased to 45 and 71% over all records within parity. Within parity by level of HAVSC, estimates of S×H variance (not shown) fluctuated without trend. Meyer (17) reported S×H variances amounting to 40 to 65% of sire variances for British Friesian-Holstein heifers. Including a S×H interaction in the model consistently reduced the sire and residual variance for ML by 1 to 7% of the corresponding estimates, ignoring interaction (Table 7). Heritability estimates for ML also decreased after fitting S×H. Similar reductions in genetic parameters were reported by others (17, 27).

Proportion of the total phenotypic variance due to S×H variance (c^2) is in Table 8 for SC and ML. For SC these estimates were below 2% in all parities. Within parity by level of HAVSC, c^2 ranged from .61 to 3.13%. Al-

TABLE 9. Product-moment (PM) correlation coefficients between sire effects for somatic cell concentrations (SC) estimated in different levels of herd average somatic cell concentration (HAVSC), 95% confidence intervals (CI), approximate limits of expectations (EXP) of PM, and genetic correlations (r_g) given by the ratio PM:EXP.

SC	PM	CI	EXP	r_g
First parity				
Q1/Q2[1,2]	.50	.44 – .55	.48 – .52	.96 – 1.04
Q1/Q3	.57	.52 – .62	.45 – .51	1.12 – 1.27
Q1/Q4	.42	.36 – .48	.41 – .48	.88 – 1.02
Q2/Q3	.53	.48 – .58	.46 – .52	1.02 – 1.15
Q2/Q4	.50	.44 – .55	.43 – .49	1.02 – 1.16
Q3/Q4	.54	.49 – .59	.41 – .48	1.13 – 1.32
Second parity				
Q1/Q2[3]	.48	.42 – .54	.40 – .47	1.02 – 1.20
Q1/Q3	.48	.42 – .54	.40 – .47	1.02 – 1.20
Q1/Q4	.41	.34 – .47	.37 – .45	.91 – 1.11
Q2/Q3	.55	.49 – .60	.40 – .47	1.17 – 1.38
Q2/Q4	.49	.43 – .55	.38 – .46	1.07 – 1.29
Q3/Q4	.55	.49 – .60	.39 – .47	1.17 – 1.41
Third parity				
Q1/Q2[4]	.39	.32 – .46	.36 – .44	.89 – 1.08
Q1/Q3	.44	.37 – .51	.33 – .43	1.02 – 1.33
Q1/Q4	.33	.25 – .40	.28 – .40	.83 – 1.18
Q2/Q3	.35	.27 – .42	.33 – .43	.81 – 1.06
Q2/Q4	.37	.30 – .44	.28 – .41	.90 – 1.32
Q3/Q4	.40	.33 – .47	.28 – .40	1.00 – 1.43

[1]The HAVSC stratification from low (Q1) to high (Q4).

[2]First parity Q1: ≤ 2.30; Q2: 2.31 to 2.64; Q3: 2.65 to 3.02; Q4: > 3.02.

[3]Second parity Q1: ≤ 2.37; Q2: 2.38 to 2.81; Q3: 2.82 to 3.28; Q4: > 3.28.

[4]Third parity Q1: ≤ 2.64; Q2: 2.65 to 3.14; Q3: 3.15 to 3.64; Q4: > 3.64.

TABLE 10. Product-moment (PM) correlation coefficients between sire effects for 305-d milk (ML) estimated in different levels of herd average somatic cell concentrations (HAVSC), 95% confidence intervals (CI), approximate limits of expectations (EXP) of PM, and genetic correlations (r_g) given by the ratio PM:EXP.

ML	PM	CI	EXP	r_g
First parity				
Q1/Q2[1,2]	.55	.49 – .59	.56 – .59	.93 – .98
Q1/Q3	.57	.52 – .62	.54 – .57	1.00 – 1.06
Q1/Q4	.54	.49 – .59	.53 – .57	.95 – 1.02
Q2/Q3	.60	.55 – .65	.54 – .58	1.03 – 1.11
Q2/Q4	.54	.49 – .59	.54 – .58	.93 – 1.00
Q3/Q4	.55	.49 – .59	.52 – .56	.98 – 1.06
Second parity				
Q1/Q2[3]	.53	.47 – .58	.44 – .49	1.08 – 1.20
Q1/Q3	.46	.39 – .52	.41 – .47	.98 – 1.12
Q1/Q4	.48	.42 – .54	.43 – .49	.98 – 1.12
Q2/Q3	.53	.47 – .58	.43 – .48	1.10 – 1.23
Q2/Q4	.54	.48 – .59	.44 – .50	1.08 – .122
Q3/Q4	.44	.37 – .50	.42 – .48	.92 – 1.05
Third parity				
Q1/Q2[4]	.50	.43 – .56	.37 – .46	1.09 – 1.35
Q1/Q3	.43	.36 – .50	.39 – .47	.91 – 1.10
Q1/Q4	.45	.38 – .51	.37 – .46	.98 – 1.22
Q2/Q3	.47	.40 – .54	.38 – .46	1.02 – 1.24
Q2/Q4	.46	.39 – .53	.37 – .47	1.02 – 1.24
Q3/Q4	.39	.32 – .46	.40 – .46	.85 – .98

[1]The HAVSC stratification from low (Q1) to high (Q4).

[2]First parity Q1: ≤ 2.30; Q2: 2.31 to 2.64; Q3: 2.65 to 3.02; Q4: > 3.02.

[3]Second parity Q1: ≤ 2.37; Q2: 2.38 to 2.81; Q3: 2.82 to 3.28; Q4: > 3.28.

[4]Third parity Q1: ≤ 2.64; Q2: 2.65 to 3.14; Q3: 3.15 to 3.64; Q4: > 3.64.

though there were differences in c^2 estimates by level of HAVSC, they showed no trend and they were nonsignificant due to large standard errors associated with them. For ML, c^2 estimates over all records within parity were a little higher than for SC. Within parity by level of HAVSC, they ranged from 1.31 and 7.33% and were in agreement with estimates of others (17, 26, 27).

Product-moment correlations between levels of HAVSC for sire effects under Model [1], and approximate limits of expected correlations, are in Tables 9 and 10 for SC and ML. Confidence intervals for the observed correlations using Fisher's log transformation are included. Estimates of genetic correlation (r_g) for expression of the same genotype in two environments were obtained by the ratio of observed to expected correlations and are also presented in Tables 9 and 10. Lower limits of r_g between SCC performance in different environments were above .80, indicating little G×E. For SC, lowest values of r_g were between the lowest and highest HAVSC subset (Q1 and Q4) in parities 1 and 2 (Table 9). High estimates of r_g for ML (Table 10) demonstrate the similarity of sire effects for ML between herds with different average SCC. These estimates of r_g are sensitive to the effective number of daughters per sire. In the present study, the average effective number of daughters in each subset ranged from 22.3 to 24.5 in first, from 15.9 to 17.8 in second, and from 10.7 to 12.7 in third parity. Sampling error due to the small average effective number of daughters may have caused some estimates of r_g to be larger than unity, especially in second and third parities.

Phenotypic and Genetic Correlation Between Traits

Estimates of phenotypic (r_p) and genetic correlations (r_g) between SC and ML by level of HAVSC within parity are in Table 11. Estimates of r_p were always negative, more so in second and third parity than in first. This was an expected result, because several reports have

TABLE 11. Phenotypic and genetic correlations between somatic cell concentration (SC) and 305-d milk (ML), by level of herd average somatic cell concentration (HAVSC) within parity.

HAVSC[1]	Phenotypic correlation	Genetic correlation
First parity		
Q1 ≤ 2.30	−.04	.20 (.10)[2]
Q2 2.31 to 2.64	−.04	.31 (.09)
Q3 2.65 to 3.02	−.05	.31 (.10)
Q4 > 3.02	−.08	.24 (.11)
Total	−.05	.24 (.06)
Second parity		
Q1 ≤ 2.37	−.14	−.06 (.14)
Q2 2.38 to 2.81	−.16	−.17 (.13)
Q3 2.82 to 3.28	−.17	−.11 (.14)
Q4 > 3.28	−.17	.12 (.14)
Total	−.16	−.17 (.07)
Third parity		
Q1 ≤ 2.64	−.16	−.29 (.16)
Q2 2.65 to 3.14	−.15	−.12 (.17)
Q3 3.15 to 3.64	−.16	−.08 (.17)
Q4 > 3.64	−.19	−.17 (.20)
Total	−.16	−.12 (.09)

[1]The HAVSC stratification from low (Q1) to high (Q4).

[2]Approximate standard errors in parentheses.

already shown a similar decline in milk production with increasing SCC (9, 18). Estimates of r_g were positive in first and negative in later parities. Positive r_g reflect an antagonistic relationship between milk yield and SCC, meaning that genetically high milk producers have a tendency toward higher SCC and greater susceptibility to mastitis. Similar results in first parity have been reported (7, 9, 11). Negative estimates of r_g between ML and SC in later parities have been observed (9, 19, 21). A possible explanation for this change is that different genetic factors may influence milk and SCC in first and later parities. Also culling in first parity based on milk yield, mastitis, or both may influence the correlation in later parities. Culling practices would remove low milk producers or potentially high milk producers with mastitis infection and high SCC. Consequently, high milk producers with low SCC would be favored to have second and later parities. Within parity, there were not substantial differences between r_g estimates obtained in different HAVSC subsets.

Genetic Correlation Among Parities

Product-moment correlations between sire effects for SC and ML in different parities and their 95% confidence intervals are in Table 12. Approximate limits of expected values of the correlation coefficients and of estimates of r_g between parities are also given. Genetic correlations of first with later parities were moderately high for SC ranging from .71 to .81, whereas between second and third parity were around unity. Comparative results from the literature for SCC measures are quite contradictory. Shook et al. (23) estimated r_g between measures of lactation SCC in pairs of the first five parities by simultaneously obtaining estimates of variance and covariance components. They reported r_g between adjacent parities ranging between .44 and .77 and averaging .55. Monardes and Hayes (20), however, estimated r_g between measures of lactation SCC in pairs of the first three parities between .90 and .97. For ML, r_g estimates of first with later parities were between .77 and .86. Between second and

TABLE 12. Product-moment (PM) correlation coefficients between sire effects for somatic cell concentrations (SC) and 305-d milk (ML) estimated in different parities, 95% confidence intervals (CI), approximate limits of expectations (EXP) of PM, and genetic correlations (r_g) given by the ratio PM:EXP.

	PM	CI	EXP	r_g
SC				
P1/P2[1]	.39	.33 − .45	.50 − .55	.71 − .78
P1/P3	.39	.32 − .45	.48 − .54	.72 − .81
P2/P3	.52	.47 − .57	.44 − .50	1.04 − 1.18
ML				
P1/P2	.51	.46 − .56	.59 − .62	.82 − .86
P1/P3	.46	.40 − .51	.55 − .60	.77 − .84
P2/P3	.56	.51 − .60	.51 − .56	1.00 − 1.10

[1]P1 = First parity; P2 = second parity; P3 = third parity.

third parity, correlations were around unity. Similarly, Maijala and Hanna (14), in a review of the literature, reported genetic correlations for milk yield between first and later parities of .80 to .85 and between second and third of .91 to 1.00. They concluded that milk yield may be a somewhat different trait in later parities than in first.

A possible explanation for the less than perfect r_g of first with later parities observed in the present study is culling based on first parity records, on both milk yield and mastitis. It has been shown that culling reduces correlations between sire evaluations for milk on first and second parity records, under mixed models (12), and modified contemporary comparisons (3). Another explanation is that different sets of genes may influence a trait in first and later parities. If SCC in first and later parities are two correlated but different traits, sire evaluation and selection based only on first parity records may not be the most effective scheme in reducing SCC and mastitis in later parities, although such a strategy would decrease generation interval. Selecting on first parity SCC records, however, to reduce overall SCC is expected to be as effective as selecting on first parity milk yield to increase overall milk yield. Therefore, if the goal is to improve resistance to mastitis and decrease the frequency of the disease across the entire productive life, sire evaluations based on progeny records from all parities should be the method of choice.

Total Somatic Cell Production

Phenotypic and r_g between SC and LT were above .95, indicating that both SCC traits are influenced by nearly the same genetic and environmental factors. Estimates of r_p between LT and ML were around .15 in first parity and .05 in later parities. Estimates of r_g between LT and ML were around .50 in first parity and .15 in later parities. These values are much higher than estimates of SC with ML due to a part-whole relationship between LT and ML. Because of its part-whole relationship with milk yield, LT is less desirable than SC for selection. Results for LT regarding estimation of h^2, r_g between parities, and studies of G×E were substantially the same as those for SC.

CONCLUSIONS

Heritability estimates of SC and LT did not vary considerably across levels of HAVSC, except at highest HAVSC and, consequently, high mastitis incidence. In these herds, which represented approximately one quarter of all data, variance among sires and heritability consistently declined, but differences from the remaining herds were small compared with SE of the estimates. Therefore, sire evaluation based on daughter performance across all herds will sufficiently predict response to selection in the general population.

Sire effects for both measures of milk SCC and lactation milk yield were consistent across herds with different average milk SCC. Genetic correlations between genotypes of the same sires evaluated in different herd levels of SCC were around unity, showing that reranking of sires across environments could be attributed to the random error associated with their estimated transmitting ability. The proportion of total phenotypic variance accounted for by S×H variance was generally low but might have been subject to a small negative bias. Sire by herd interaction reflects the similarity between daughters of a sire in the same herd, and represents both G×E and covariances between the records of half-sisters herdmates. Failure to account for this interaction will cause an overestimation of the accuracy of sire evaluation, even though reranking of sires may not be affected considerably, as shown in the present study. The impact of this bias becomes more severe in proofs of sires whose daughters are located in one or very few herds. In data used in this study, on the average only about 10% of sires had daughters with records in less than 5 herds. However, these were somewhat selected data sets because of the specific edits applied. The proportion of these sires in the entire sire population may determine the necessity of incorporating c^2 effects in a national sire evaluation scheme.

There was evidence of change in variation of SCC from first to later parities. This was indicated by changes in the r_g of both SCC traits with milk yield. Also r_g between first and later parities was less than unity for SCC. Somatic cell count in young and mature ages may be two different but correlated traits. Genetic correlations between second and third parity were around unity, meaning that the same genetic factors influence SCC in these two parities. Results were similar for milk yield.

Sire evaluation and selection on first parity records would avoid bias due to culling and decrease the generation interval. Such practice,

however, may not necessarily result in the most efficient reduction of SCC in later parities. Also, records from later parities may be better indicators of resistance to mastitis, because of more frequent mastitis occurrence, than first parity records. Sire evaluation for SCC should be based on progeny records in all parities. This approach will also increase the accuracy of sire evaluations.

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REFERENCES

1 Ali, A.K.A., and G. E. Shook. 1980. An optimum transformation for somatic cell concentration in milk. J. Dairy Sci. 63:487.
2 Calo, L. L., R. E. McDowell, L. D. Van Vleck, and P. D. Miller. 1973. Genetic aspects of beef production among Holstein-Friesians pedigree selected for milk production. J. Anim. Sci. 37:676.
3 Cassell, B. G., B. T. McDaniel, and H. D. Norman. 1983. Impact of culling on modified contemporary comparisons sire evaluations. J. Dairy Sci. 66:1359.
4 Coffey, E. M., W. E. Vinson, and R. E. Pearson. 1986. Potential of somatic cell concentration in milk as a sire selection criterion to reduce mastitis in dairy cattle. J. Dairy Sci. 69:2163.
5 Dabdoub, S.A.M., and G. E. Shook. 1984. Phenotypic relationships among milk yield, somatic cell count, and clinical mastitis. J. Dairy Sci. 67(Suppl. 1):163. (Abstr.)
6 Danell, B. 1982. Interaction between genotype and environment in sire evaluation for milk production. Acta Agric. Scand. 32:33.
7 Emanuelson, U., B. Danell, and J. Philipsson. 1988. Genetic parameters for clinical mastitis, somatic cell counts, and milk production estimated by multiple-trait restricted maximum likelihood. J. Dairy Sci. 71:467.
8 Grootenhuis, G. 1981. Mastitis prevention by selection of sires. Vet. Rec. 108:258.
9 Heuven, H.C.M., H. Bovenhuis, and R. D. Politiek. 1988. Inheritance of monthly somatic cell count and its genetic relationship with milk yield. Livest. Prod. Sci. 18:115.
10 Hickman, C. G., A. L. Lee, and K. Gravir. 1969. Genotype × season × method interaction in evaluating dairy sires from progeny records. Can. J. Anim. Sci. 49: 151.
11 Kennedy, B. W., M. S. Sethar, J. E. Moxley, and B. R. Downey. 1982. Heritability of somatic cell count and its relationship with milk yield and composition in Holsteins. J. Dairy Sci. 65:843.
12 Lofgren, D. L., B. G. Casell, H. D. Norman, and B. T. McDaniel. 1983. Effects of culling on sire evaluations by mixed models. J. Dairy Sci. 66:2418.
13 Lush, J. L. 1950. Inheritance of susceptibility to mastitis. J. Dairy Sci. 33:121.
14 Maijala, K., and M. Hanna. 1974. Reliable phenotypic and genetic parameters in dairy cattle. Proc. 1st World Congr. Genet. Appl. Livest. Prod. 1:541.
15 Meyer, K. 1985. Maximum likelihood estimation of variance components for a multivariate mixed model with equal design matrices. Biometrics 41:153.
16 Meyer, K. 1986. Restricted maximum likelihood estimation of variance components–in practice. Proc. 3rd World Congr. Genet. Appl. Livest. Prod. 12:454.
17 Meyer, K. 1987. Estimates of variances due to sire × herd interactions and environmental covariances between paternal half-sibs for first lactation dairy production. Livest. Prod. Sci. 17:95.
18 Miller, R. H., U. Emanuelson, E. Persson, L. Brolund, J. Philipsson, and H. Funke. 1983. Relationships of milk somatic cell counts to daily milk yield and composition. Acta Agric. Scand. 33:209.
19 Monardes, H. G., and J. F. Hayes. 1985. Genetic and phenotypic relationships between lactation cell counts and milk yield and composition of Holstein cows. J. Dairy Sci. 68:1250.
20 Monardes, H. G., and J. F. Hayes. 1985. Genetic and phenotypic statistics of lactation cell counts in different lactations of Holstein cows. J. Dairy Sci. 68:1449.
21 Monardes, H. G., J. F. Hayes, and J. E. Moxley. 1984. Heritability of lactation cell count measures and their relationships with milk yield and composition in Ayrshire cows. J. Dairy Sci. 67:2429.
22 Shook, G. E. 1986. Genetic aspects of mastitis. Proc. 25th Annu. Mtg. Natl. Mastitis Counc. Arlington, VA.
23 Shook, G. E., F. Ruvuna, and A.K.A. Ali. 1982. Genetic parameters of lactation average of somatic cell concentration in milk. Proc. 2nd World Congr. Genet. Appl. Livest. Prod. 8:142.
24 Smith, S. P., and H. U. Graser. 1986. Estimating variance components in a class of models by restricted maximum likelihood. J. Dairy Sci. 69:1156.
25 Strandberg, E., and G. E. Shook. 1989. Genetic and economic responses to breeding programs that consider mastitis. J. Dairy Sci. 72:2136.
26 Syrstad, O. 1976. Interaction between genotype and nutrition in dairy production – a review. World. Rev. Anim. Prod. 12:33.
27 Tong, A.K.W., B. W. Kennedy, and J. E. Moxley. 1977. Sire by herd interactions for milk yield and composition traits. Can. J. Anim. Sci. 57:383.
28 VanRaden, P. M., and A. E. Freeman. 1985. Rapid method to obtain bounds on accuracies and prediction error variances in mixed models. J. Dairy Sci. 68:2123.
29 Vecht, U., G. E. Shook, R. D. Politiek, G. Grootenhuis, W. J. Koops, and D. G. Groothuis. 1985. Effect of bull selection on somatic cell count in first lactation on cell counts and pathogens in later lactations. J. Dairy Sci. 68: 2995.
30 Wiggans, G. R., and G. E. Shook. 1987. A lactation measure of somatic cell count. J. Dairy Sci. 70:2666.